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Cardiac Arrhythmias as Related to Anesthesia

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Doctor of Medicine

College of Medicine, University of Nebraska

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Omaha, Nebraska

## CARDIAC ARRHYTHMIAS AS RELATED TO ANESTHESIA

### I. Types of Arrhythmias

- A. Characterized by a Rapid Regular Pulse
- B. Characterized by a Slow Regular Pulse
- C. Characterized by an Irregular Pulse

### II. Incidence

- A. Pre-operative Cardiac Status
- B. Age and Sex
- C. Agents

- 1. Cyclopropane
- 2. Ether
- 3. Nitrous Oxide
- 4. Fluothane
- 5. Chloroform
- 6. Trilene
- 7. Pentothal
- 8. Muscle Relaxants
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### III. Other Factors during Anesthesia

- A. Hypoxia
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- C. Reflexes
- D. Hypothermia
- E. Cardiac Arrest

### IV. Treatment or Reversal

Let us first examine the various types of arrhythmias. I will classify them according to rate and rhythm rather than one based upon abnormal sites of impulse formation or conduction pathways:

I. Arrhythmias Characterized by a Rapid Regular Pulse

- A. Sinus Tachycardia
- B. Supraventricular Tachycardia
- C. Atrial Flutter
- D. Ventricular Paroxysmal Tachycardia

II. Arrhythmias Characterized by a Slow Regular Pulse

- A. Sinus Bradycardia
- B. Atrio-ventricular Nodal Rhythm
- C. Auricular Standstill with Auriculo-Ventricular Nodal Escape
- D. Incomplete Heart Block
- E. Complete Heart Block

III. Arrhythmias Characterized by an Irregular Pulse

- A. Sinus Arrhythmia
- B. Sino-Auricular Block
- C. Premature Contractions
  - 1. Atrial Premature Contraction
  - 2. Nodal Premature Contraction
  - 3. Ventricular Premature Contraction
- D. Atrial Fibrillation
- E. Ventricular Fibrillation

## F. Second Degree A-V Block (Wenckebach Phenomenon)

Sinus tachycardia is considered by most authorities as a rate of greater than 100 beats per minute. Usually this does not exceed 150 beats per minute in adults but may be 180 or higher. Diagnosis usually is rapid, regular pulse, usually less than 150, which alters in rate and regularity with respiration. It is frequently accompanied by other clinical signs and symptoms such as pallor and fever. Usually this tachycardia is a result of increased sympathetic tone or decreased vagal tone. Perhaps the three most common causes are hypoxia, pain, and hypovolemia due to blood loss. Drugs such as atropine and meperidine are a common etiology. Hyperthermia, extreme toxicity, burns, thyrotoxicosis, and early congestive heart failure are other etiologies. Supraventricular Paroxysmal tachycardias are characterized by an abrupt onset and usually greater rapidity (140-220 beats/minute) which distinguishes them from sinus tachycardia. Rhythm is almost always regular. Common causes are digitalis toxicity, quinidine overdosage, congestive heart failure, thyrotoxicosis, and hypoxia. Atrial flutter is the result of rapid formation of ectopic auricular stimuli at a rate of 200-380 beats per minute. However, since only a percentage of these auricular impulses are transmitted to the ventricles, the radial pulse has a rate of 70-160 beats per minute. The radial pulse is dependent upon the degree of block present. Carotid sinus pressure is a helpful diagnostic aid in flutter with a rapid ventricular rate. The rate will slow markedly but increases again when pressure is released. In

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contrast, this compression will usually permanently convert an atrial or nodal paroxysmal tachycardia to sinus rhythm with no effect on a ventricular tachycardia or auricular fibrillation. In a slow ventricular rate, rapid auricular rate can usually be observed in the jugular vein pulsations. In flutter, the ventricular rate is usually regular, which helps to differentiate this condition from auricular fibrillation. Auricular flutter is usually encountered more frequently over the age of sixty. Atrial flutter has two serious prognostic implications: (1) it is usually superimposed upon a diseased heart and, (2) it frequently causes congestive heart failure. The latter is heightened when a rapid ventricular rate is present. Ventricular paroxysmal tachycardia is a rare arrhythmia. It is almost always imposed upon a seriously damaged heart. The pulse rate is usually between 140-180 and there is usually a detectable irregularity in rhythm which is slight. This arrhythmia is unaffected by carotid sinus pressure.

Sinus bradycardia is characterized by a pulse rate of 60/minute or less in an adult. This varies in a child according to age. The rhythm may at times be regular but more often there is an associated sinus arrhythmia. Parasympathetic drugs, spinal anesthesia, and carotid sinus pressure are some of the etiologic factors. A-V Nodal Rhythm occurs when the normal sinus pacemaker is sufficiently depressed. A slow pulse rate may be the only clinical sign but a pronounced jugular vein thrust is characteristic and attributable to a regurgitant filling of the neck veins when the auricle contracts against a closed tricuspid valve. This is usually a temporary

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disturbance caused by vagal inhibition of the sinus node. Atrial standstill with nodal rhythm is extremely rare. In contrast to A-V nodal rhythm, auricular beats do not occur. It should be considered whenever a slow pulse is present. Incomplete A-V Block is caused by organic disturbances of the conduction pathway. The A-V node fails to respond to sinus stimuli at regular intervals and only every second, third, and fourth auricular impulse reaches the ventricle. The pulse is slow and regular. The neck veins show small auricular pulsations which occur two, three, or four times more frequently than the radial pulse. Complete A-V Block usually has a ventricular rhythm between 30-60/ minute. The rhythm is extremely slow, forceful and regular. Here again, observation of the neck vein pulsations will indicate the presence of independent auricular activity at a faster rate.

Sinus Arrhythmia is commonly present in children and young adults, due to a disturbance in the rhythmic production of impulses at the sino-auricular node. The irregularity is associated with the phases of respiration---inspiration usually increasing the heart rate, due to decreasing vagal tone. Sino-Auricular Block is characterized by a sudden standstill of the entire heart for one or more beats. Single beats may drop out in regular sequence or runs of two or three dropped beats may occur. Clinically, a regular pulse is suddenly interrupted by a long pause, after which the regularity is resumed. S-A block is due to increased vagal tone, with resultant depression of the sinus node impulse formation. Premature

Contractions constitute the most common cause of pulse irregularity. This occurs when the atria or ventricles are stimulated before the next regular sinus impulse is due. The origin of the premature contractions may be in the auricles, the A-V node, or the ventricles; and they may occur in regular sequence or infrequently. They differ from S-A block in having a compensatory pause, due to the failure of the ventricles to respond to the next normal sinus impulse. Premature beats can be distinguished from sinus arrhythmia by noting the phase of respiration. The cause seems to be a manifestation of localized irritability of the heart, and are a common finding in patients over 50 years of age. If they occur with more than occasional frequency, either serious heart disease or toxicity is usually present. Atrial Fibrillation can usually be diagnosed clinically by the complete irregularity of the rate, rhythm, and force of the heart beat felt in the radial pulse, and by the pulse deficit observed by simultaneous auscultation of the apex beat and palpation of the pulse. The neck veins show irregular pulsations. Early untreated auricular fibrillation is usually characterized by a rapid ventricular rate. The causes are numerous. Ventricular fibrillation presents a very irregular rapid rate and produces sudden death. Blood pressure falls to zero and heart sounds become inaudible. Pulse rate is usually greater than 300 beats per minute, and the EKG is diagnostic with undulating waves. The causes are usually due to coronary occlusion or toxicity to the heart. Lastly, Incomplete A-V Block (Wenckebach Phenomenon) produces an irregular pulse, due to each successive stimulus leaving



the auricles finding an increased difficulty in transversing the A-V node; finally the impulse is not transmitted at all, and ventricular contraction fails to occur. This cycle repeats itself, resulting in a series of dropped beats, usually occurring at regular intervals.

Atrial-ventricular dissociation was the most frequently observed arrhythmia as seen and recorded in a study by Dodd, Sims, and Bone.<sup>2</sup> Following in decreasing order were ventricular premature contractions, atrial premature contractions, atrial-ventricular block, sinus arrhythmia, and atrial fibrillation. Transient bradycardia, and tachycardias may often occur too.

Cannard and his group at Pennsylvania<sup>1</sup> also state that extrasystoles are the most commonly observed arrhythmia in anesthesia and surgery. As opposed to those seen medically, most extrasystoles seen during anesthesia can be easily eliminated by removing the cause. Arrhythmias are more likely to be observed during halothane, chloroform, cyclopropane, or ethyl chloride than other types of anesthesia. Extrasystoles are usually insignificant and important only when frequent enough to produce hypotension. The etiology is numerous and includes drugs, hypothermia, metabolic imbalance, and decreased oxygen perfusion.

Tachycardia is generally well tolerated in normal hearts and is more likely to cause damage in "diseased" hearts. Ventricular tachycardia is more dangerous as it may lead to hypoxia and fibrillation. As in all other arrhythmias, when occurring during

anesthesia, if the cause is found, tachycardia can usually be eliminated by removing the cause. Usually there is spontaneous disappearance with increased oxygen saturation of blood or decreased anesthesia.

According to Dodd, 51.4% of his heart disease patients (hypertension, murmurs, history of myocardial disease etc.) had some arrhythmia during anesthesia, whereas only 19.9% of the non-heart disease patients did. Also, the more dangerous arrhythmias occurred more frequently in the former group.

Studies have indicated that there is no direct correlation between the incidence of arrhythmias and age groups.<sup>8</sup> However, because heart disease is more prevalent in older age groups, and arrhythmias are more prevalent in heart disease patients, the incidence of arrhythmias is greater in older age groups. Likewise, sex was not directly related to incidence.

One study has determined the following incidence of arrhythmias according to the following agents.<sup>2</sup> Recorded only are the more commonly used agents:

1. Cyclopropane---50%
2. Halothane       44%
3. Ether           38%
4. Nitrous Oxide 33.3%
5. Spinal          31.3%
6. Thiopental     9.8%

What do the above figures actually relate? One must realize that such agents as thiopental are mostly used for inductions whereas

ether, cyclopropane, and halothane are often used for longer operations. Also, nitrous oxide, is often combined with other agents during surgery, as with the three named above. Inhalation agents cause more arrhythmias than intravenous ones. Also, when arrhythmias occurred during ether anesthesia, they were more often supraventricular than with other agents. Now let us examine individual anesthetics somewhat more closely.

Cyclopropane causes a gradual slowing of the heart beat. If a bradycardia should develop, atropine may block this. Inadequate atropinization may lead to a degree of heart block. The main arrhythmias recorded have been premature ventricular contractions and multifocal ventricular tachycardia.<sup>4</sup> Carbon dioxide enhances these arrhythmias. Rapid lowering of mean cyclopropane arterial blood saturation increases ventricular arrhythmias. Intravenous atropine should not be given in doses greater than 0.2 mg. at a time due to the increased incidence of arrhythmias with a higher concentration of atropine used with cyclopropane at one injection. Robins states that cardiac irregularities are easily produced in cats respiring a mixture of 30-40% cyclopropane in oxygen.<sup>7</sup> The rhythm can be returned to normal by increasing the oxygen level in the blood. An intact sympathetic nervous system isn't needed to produce these arrhythmias. The level of  $P_{CO_2}$  is the most important factor in producing arrhythmias when there is a fixed level of anesthesia with cyclopropane. Robins also found the  $P_{CO_2}$  to be one-third lower during regular rhythm than during arrhythmia.

These irregular rhythms during cyclopropane and high  $P_{CO_2}$  can be returned to normal by the intravenous injection of amobarbital or secobarbital (5-8 mg./kg.) Cyclopropane enhances the response of cardiac myocardium to catecholamines.

Cyclopropane also, without prior premedication, causes a marked cardiac output during light anesthesia, but as concentration increases, reduced output occurs. There is no obvious relation between cardiac rate and depth of anesthesia. In a study by Jones, ventricular extrasystoles occurred on eleven occasions in eight subjects but all but one was apparently initiated by mechanical stimulation---tracheal intubation, coughing, etc.<sup>16</sup> In most cases the arrhythmias were transient, lasting a few seconds to a few minutes.

In laboratory animals anesthetized with ether, any developing arrhythmias are supraventricular, until the heart begins to fail from overdose. Ether has been used to abolish cyclopropane induced arrhythmia. In man, the majority of arrhythmias produced are supraventricular, including atrial and nodal extrasystoles, nodal rhythm, and pacemaker displacement. The ethers appear to have the greatest antiarrhythmic activity of all currently used general anesthetics. In fact, ether is often added to other anesthetics or completely substituted if arrhythmia develops.

Nitrous oxide, intravenous barbiturates, and spinal anesthetics are relatively free of arrhythmias as a major problem. When arrhythmias do occur, they are usually due to excessive concentration or physiological unsafe oxygen levels.

Fluothane usually causes a bradycardia with increased concentration of the drug, and hypotension and arrhythmias are also prevalent. Increased ventricular excitability, including bigeminy, multifocal ventricular extrasystoles, and multifocal ventricular tachycardia are most frequently observed with fluothane. The degree of hypercarbia required to initiate arrhythmia is not related to the inhaled concentration of fluothane. In a closed circuit, where there is a greater degree of carbon dioxide retention, fewer ventricular irregularities occur. Halogenated hydrocarbons, of which fluothane is a member, cause increased sensitization to epinephrine.

In man, a bradycardia occurs during induction and maintenance with chloroform. Pre-operative atropine helps to reduce cardiac irregularities. However, the incidence of ventricular irregularities is high---Hill observed premature ventricular contractions in 50% of a small series. Although incidence of ventricular arrhythmias may be high, there are few documented deaths from ventricular fibrillation. The greatest danger of arrhythmia occurs during passage from deep to light anesthesia. Chloroform may cause cardiac arrest in three ways: vagal inhibition, ventricular fibrillation, or direct myocardial depression. If irregularities occur in the early induction stage, it is probably due to vagal inhibition, as an increase in baroreceptor sensitivity. Therefore full therapeutic doses of vagal depressants should be used prior to chloroform induction and the potentially dangerous induction stage accomplished with intravenous thiobarbiturate. Cardiac sensitivity is probably no greater than in cyclopropane or trilene

anesthesia.

Barnes and Ives found electrocardiographic changes in 33 out of 40 patients undergoing trilene anesthesia. In light anesthesia bradycardia and shift of pacemaker towards a nodal rhythm were most prevalent. In deeper anesthesia, ventricular extrasystoles and multifocal ventricular tachycardia occurred in some cases. They believed trichorethylene should not be used as an adjunct to nitrous oxide, especially if adrenalin was used during the operation. Therefore, potentiation of serious arrhythmias in trilene anesthesia is enhanced with the use of adrenalin and similar sympathomimetic agents.

Gruber first reported that cardiac arrhythmia, mainly premature ventricular contractions, was caused by the administration of thiopental sodium.<sup>5</sup> He attributed the sulfur ion in the thiopental as causing increased irritability of the ventricles so that they would respond more quickly to weaker impulses. Two theories have been expounded in the literature to explain the etiology of cardiac arrhythmia in thiobarbiturate anesthesia. One states that anoxia produced by a decrease in respiration causes the change in cardiac rate and rhythm. The other explains the arrhythmia as being due to the sulfur causing increased excitability of the ventricles so they respond more quickly to muscle stretch, leading to PVCs when the blood pressure remains approximately the same or is increased. Frenz and Benitz, in their investigation of a number of barbiturates and thiobarbiturates on the heart and

blood pressure, observed cardiac arrhythmias with the use of thiobarbiturates but never with the use of barbiturates. The cardiac arrhythmias were noted only when the blood pressure increased. Gruber has shown that anoxia caused by thiobarbiturates anesthesia probably plays no role in the production of cardiac arrhythmias by comparing the results his experiments produced with those of anoxia produced by the inhalation of nitrogen. Conway believed that the level of blood pressure was the sole agent responsible for the production of ventricular arrhythmias. He felt the level of blood pressure acted directly upon the excitability of heart muscle either through excessive tension developed during systole or by influencing diastolic size. The cardiac arrhythmias caused by thiobarbiturates are rather simple harmless ones with anesthetic doses. Oxygen should be administered, however, to counteract any possible harmful effects of anoxia on the CNS and other systems rather than upon the heart.

Since succinylcholine and acetylcholine have similar structures, the former causes bradycardia, and also shift of the pacemaker to the atrio-ventricular node on occasion. Relaxants of the curare type appear to have little effect upon producing arrhythmias, providing adequate ventilation is maintained.<sup>3</sup> The bradycardia, according to Turndorf and Dripps, occurs with intravenous, but not with intramuscular, succinylcholine. The bradycardias and arrhythmias appear less if a belladonna preanesthetic medication is given, however, morphine increases the likelihood of bradycardia.

Vasopressors are usually added to local anesthetics to prolong

their effect or used to raise or maintain blood pressure. Three classes of pressor drugs according to Aviado are thus:

(a) Those which increase activity of the sino-atrial node and the initiation of ventricular arrhythmia. These include epinephrine, ephedrine, and norepinephrine.

(b) Those which produce sino-atrial bradycardia and uncertain effects on ventricular foci. Drugs include vasoxyl and neo-synephrine.

(c) Those which produce sino-atrial tachycardia and a decrease of ventricular arrhythmia. Wyamine is a member of this group, although this drug may produce arrhythmias.

Epinephrine has a tendency to overstimulate the heart leading to irregularities in the conducting system such as ventricular fibrillation. Norepinephrine causes consistent arrhythmias in dogs and variable effects in man. Vasoxyl produces bradycardia in man which can be blocked by atropine. The bradycardia is due to vagal stimulation via the baroreceptors and slowing of the atrial-ventricular conduction. Wyamine is the only pressor agent which stimulates the sino-atrial node and yet may stop or prevent experimental arrhythmias.

Mild to moderate hypoxia causes tachycardia and increased blood pressure whereas more severe hypoxia causes bradycardia and a decreased blood pressure. The electrocardiogram is a poor indication of hypoxia in the healthy human. Sinus arrhythmia, wandering pacemaker, paradoxical atrial tachycardia, and premature atrial contraction are seen in prolonged hypoxia. It is well known that heart



damage can easily occur secondary to decreased oxygen supply. If reperfusion does not occur until after 15 minutes, immediate reversal isn't always possible. The pacemakers and conducting system can tolerate several degrees of oxygen lack, the atrial-ventricular node having the least resistance. However, the myocardium is much less resistant.

Hypercarbia causes increased release of catechol amines from sympathetic nerve endings in the myocardium, especially with cyclopropane anesthesia. Carbon dioxide overdose causes among other things, tachycardia and hypertension. But these are usually "late" signs of hypercarbia. Carbon dioxide also has an indirect depressant effect on cardiac output, causing a change in pH with a shift of potassium ion from the cells, and may lead to cardiac standstill. Because hyperkalemia occurs with increased carbon dioxide concentration, this helps to potentiate the standstill. In animal experiments, cardiac irregularities occur if a high carbon dioxide level is allowed to return to normal rapidly. These irregularities are less frequent in man during carbon dioxide accumulation. The arterial carbon dioxide threshold at which arrhythmia occurs varies with different anesthetic agents, being lower with cyclopropane and higher with halothane.

Several procedures have caused cardiac slowing or arrhythmias. They are: intubation or mechanical stimulation of the trachea in the presence of oxygen, oculo-cardiac reflex, carotid sinus reflex, traction on abdominal and pelvic organs, and irritation of the

pleura and pericardium. The majority of reflexes are parasympathetic causing bradycardia, displacement of the pacemaker, and various degrees of heart block. Many sinus tachycardias have been reported, along with premature ventricular contractions, nodal rhythm, sinus bradycardia, ventricular tachycardia, and auricular fibrillation.

When the body temperature falls below 30 degrees centigrade, the risk of ventricular fibrillation progressively increases. At 30 degrees centigrade the safe interval of circulatory arrest is only 8-10 minutes. By adding extracorporeal circulation, the safe limit has been decreased to 4 degrees centigrade and the complete cessation of circulation can last for 50 minutes. During cooling, the bradycardia which insues, is refractory to atropine or vagotomy, and conduction decreases porportionally. Arrhythmias are frequent below 28 degrees centigrade and include atrio-ventricular block, atrial fibrillation, and premature ventricular contractions. Ventricular fibrillation, of course, is the cheif hazard. Hypothermia may produce irritable foci anywhere in the heart, causing extrasystole.

Cardiac arrest or cardiac failure is the failure of effective myocardial contraction. This may result from asystole or ventricular fibrillation. Most hearts stand still in diastole; a smaller porportion in ventricular fibrillation. Cardiac arrest is observed with increased frequency in extracorporeal circulation and hypothermia. The immediate danger is irreversible cerebral damage.

Significant etiology includes hypotension, hypovolemia, anemia, asphyxia, nervous stimuli, chemical stimuli, i.e. hypoxia, hypercarbia, acidosis, hyperkalemia, and physical stimuli, i. e. cardiac tamponade, position of the patient etc. Hypoxia is the most important single factor in sudden cardiac collapse.<sup>6</sup> Cerebral anoxia of four minutes duration in a well-oxygenated patient at the time of sudden cardiac collapse results in irreversible damage to the brain. In an hypoxic patient, cerebral anoxia of one minute or less, may produce similar results.<sup>6</sup> Certain warning signs include:

- (1) sudden disappearance of radial, femoral, or carotid pulsations
- (2) unobtainable blood pressure
- (3) absence of wound bleeding
- (4) pallor of the skin or cyanosis
- (5) cessation of cardiac impulse
- (6) EKG showing asystole or ventricular fibrillation
- (7) apnea or sudden gasping respiration
- (8) dilated, fixed pupils in the later stages

The EKG can continue to show normal complexes in spite of inadequate cardiac output. The EEG is actually a more sensitive indicator, being flat in 20 seconds after complete cessation of the heart beat.

The prognosis in patients with sudden cardiac collapse is chiefly dependent on rapid restoration of an effective oxygen system and rapid restoration of the heart beat and circulation. Various procedures have been favored as the initial step following diagnosis and include:

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- (1) Pounding on the chest wall
- (2) Injecting epinephrine into the heart
- (3) Use of an external defibrillator or cardiac stimulator
- (4) Thoracotomy with manual cardiac compression
- (5) Closed chest cardiac massage
- (6) Closed chest cardiac massage with artificial respiration

On the basis of recent literature, the regime of closed chest cardiac massage with artificial respiration is the current initial therapy of choice. The first operator should maintain adequate oxygenation by endotracheal tube at a rate of 14-16 times per minute. If a tube isn't available, a mask or mouth to mouth respiration may be performed. The carotid, femoral, or radial pulse should be checked continuously. The second operator should simultaneously perform closed-chest cardiac massage, at 60-80 times per minute for at least 3-4 minutes. Adequacy of massage is verified by palpating the carotid pulsation, maintenance of small pupils, a blood pressure greater than 80 per minute, or regular respiratory pattern. If no pulse is present within four minutes, a thoracotomy and manual compression at 72 per minute should be performed. Many feel the thoracic approach for cardiac massage is still the method of choice. Supportive treatment such as calcium gluconate, epinephrine, nephen-termine, digitalization, atropine, hypertonic urea, and hypothermia may be helpful. It is suggested that the literature be reviewed regarding these later methods.

Many irregularities of anesthesia can be easily reversed.

The concentration of the inhaled agent can be reduced, oxygen can be supplied in increased percentage, hypercarbia can be reduced, as can hypoxia. Temperature can be increased or decreased and fluid and electrolytes are given so that the level of anesthesia may be proportionally decreased. Formerly intravenous atropine was given to correct atrio-ventricular dissociation, if it persisted, but at present, drugs are rarely used. When frequent ventricular extrasystoles develop, atropine (0.2 mg.) may be given intravenously. Ventricular premature contractions can also be often corrected by changing the anesthetic agent or temporarily discontinuing the operation and washing out the lungs with oxygen. Barbiturates abolish spontaneously-induced cyclopropane arrhythmias but not epinephrine-induced ones. Although diabenamine and procaine amide often lower the incidence of epinephrine-induced cardiac arrhythmias, they are seldom used due to their causing pronounced hypotension. Procaine and procaine amide have diminished a large percentage of arrhythmias caused by reflexes. If the reflex induced arrhythmias are accompanied by hypercarbia, 10% oxygen will often reduce the abnormal rhythm. Supraventricular tachycardias are generally treated with some form of vagal stimulation, such as carotid sinus pressure, or neosynephrine. Parasympathetic drugs are seldom used due to the possibility of asystole and hemiplegia. Digitalis and sympathomimetic drugs (in doses slow and low enough to prevent hypertension) may be used also. Quinidine and procaine amide should be avoided. In ventricular tachycardias, vasopressors are used to

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increase the coronary blood flow and systemic blood pressure. With atrial fibrillation and flutter, the agent of choice is digitalis, especially if hypotension is present. Atropine is the drug of choice in treating bradycardia, especially if the latter has a hypersensitivity etiology. Complete heart block responds well to atropine, 1-2 mg., especially if there is an accompanying Stokes-Adams Syndrome. Then a vasopressor, such as Isuprel, may be given in solution of 1 mg. in 200 ml. of 5% D/W. Epinephrine shouldn't be used, however. Ether and nitrous oxide are considered the best general anesthetics for this condition. Extrasystoles are usually removed by eliminating the cause but if this fails, xylocaine, quinidine, or procaine amide may be used under electrocardiogram control.

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